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THE

OPHTHALMIC REVIEW

A MONTHLY RECORD OF OPHTHALMIC SCIENCE

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TOXIC AMBLYOPIA FROM IODOFORM.

By Priestley Smith.

Ophthalmic Surgeon to the Queen's Hospital, and Lecturer on Ophthalmology in the Queen's Faculty of Medicine, Mason College, Birmingham.

In the year 1882 Hirschberg reported the case of a girl in whom a typical toxic amblyopia occurred during the after-treatment of a hip-joint operation. The only toxic substance to which it could be attributed was the iodoform with which the wound was dressed, and when this was omitted the amblyopia disappeared. (*Centralbl. f. prakt. Augenheilkunde*, 1882, p. 92.)

In 1886 Dr. E. Hutchinson recorded a case of toxic amblyopia from the continued use of iodoform and creasote in pills. The patient was a tall, well-made man, of good habits, temperate in food and drink, and did not smoke at all. He had a cough and had lost flesh. For a period of six months he had taken daily nine grains of iodoform and six grains of creasote in pills. When the pills were stopped and subcutaneous injections of strychnia begun, improvement was noticed in a few days, and about two months later vision had risen to 20-30ths and 20-40ths in the two eyes respectively. During recovery the patient resumed the use of the creasote, but took no more iodoform. (*New York Medical Journal*, Jan. 2nd, 1886.)

These are, so far as I can ascertain, the only cases on record in which toxic amblyopia has been attributed to the use of iodoform, and to judge from certain later references to these cases, they have not been regarded

as quite conclusive. The case which I now record seems to prove beyond question that iodoform is one of the toxic substances which may induce a retrobulbar neuritis characterised by slight changes at the papilla and central scotoma.

The case occurred in the medical wards of the Queen's Hospital under the care of my colleague, Dr. Foxwell, to whom I am indebted for the use of a very full clinical record made at the time. A brief summary of this will be sufficient here.

Harry B., aged 31, was admitted Nov. 18th, 1891. He had been treated three months earlier for pleuritic effusion. He was now suffering from diarrhœa, vomiting, emaciation, and rapid loss of strength. He was found to have, among other changes, dry pleurisy at both bases, and a solid substance in the abdomen which was regarded as being probably due to tuberculosis of the omentum. The diagnosis was chronic tubercular pleurisy and peritonitis.

From Dec. 5th to Jan. 15th, a period of forty-one days, he was treated with iodoform in pills, and during this time took no other drug excepting five-grain doses of bicarbonate of soda in infusion of calumba. He took at first two grains of iodoform three times a day, and the quantity was steadily increased until during the last ten days he took four grains eight times a day, *i.e.*, thirty-two *grains per diem*. He took altogether about 1,000 grains in forty-one days. The iodoform was then stopped on account of toxic symptoms, especially amblyopia.

The onset of the amblyopia was associated with headache, giddiness, and faintness, the latter being due probably to a weak heart; also with diarrhœa, a constant taste and smell of iodoform, twitching of the hands, and emotional depression; also with a marked change in the urine, viz., from high acidity with uric acid and urates, to alkalinity with triple phosphates.

On the third day after the iodoform was stopped there was great drowsiness and slight left ptosis; the next day the drowsiness had given place to irritability, and the ptosis was gone. There were numbness and tingling in the legs, and the knee jerks were increased.

Four days after the iodoform had been discontinued I examined the patient with regard to his eyes. The refraction was normal, the media were clear in both eyes. In both there was slight haziness of the disc margin, but no pronounced papillitis. Vision was greatly impaired, a well-marked central scotoma, absolute for white paper at or near the fixation point, being present in each eye. Tested with red the scotomata appeared to be larger than those commonly found in tobacco amblyopia. The loss of vision appeared to have begun a day or two before the iodoform was stopped, and whilst the patient was taking the maximum quantity of thirty-two grains a day, and to have rapidly increased from day to day. Up to this time he had certainly seen well, for he had observed the pictures on the far side of the ward and had read the newspaper as he lay in bed. Re-examined seven days after the iodoform was stopped, the haziness of the disc margins was rather more pronounced. The patient thought his vision had already begun to improve.

Four weeks later he came again under my notice and had still a central colour scotoma in both eyes, with a very small absolute scotoma just below the fixation point. Vision: R. 6-36ths, L. 6-24ths. He then received subcutaneous injections of strychnia for a week, and later iron and strychnia by the mouth. Vision improved steadily, and on April 8th, *i.e.*, about three months after the onset of the amblyopia, it had risen to 6-8ths in each eye, and no scotomata could be found. The patient's general condition was much better than at the time of his admission into hospital.

The following facts as to the patient's use of tobacco

appear to exclude that agent as a possible cause of the amblyopia. Before admission he had habitually smoked about two ounces a week; during his stay he did not smoke at all excepting about a quarter of an ounce on Christmas Day. It was, therefore, about seven weeks after the discontinuance of regular smoking, and three weeks after the one indulgence at Christmas, that the rapid onset of the amblyopia occurred. Moreover, during the period of recovery from the amblyopia, when he was no longer an in-patient, he resumed his smoking, though probably in smaller amount than before.

With regard to the internal administration of iodoform in general, Dr. Foxwell has kindly written me the following note:—"I have given iodoform to a large number of phthisical patients during the past five years, and have always endeavoured to reach a dose of thirty grains per diem, and to continue this for about six weeks. The patients have either complained of gastric disturbance when taking a small dose—and I have then generally found it useless to continue the treatment—or have submitted to the exhibition of the full dose with no bad symptom except occasionally a vague depression. Only once did I notice any cerebral affection—aphasia in a gentleman with bronchiectasis. Never, to my remembrance, has any one of my patients complained of visual defect. I have now under my care a young gentleman of 18, phthisical, who has taken twenty-seven grains daily for fourteen months, and has gained a stone in weight whilst doing so."

NOTES OF A CASE OF SUBHYALOID AND VITREOUS HÆMORRHAGE.

By J. B. Lawford.

The case, of which notes are appended, presents, I think, sufficient points of interest to merit a brief record, although it is not of much value in the elucidation of the obscurity still surrounding these cases.

Mrs. S——, aged 55, an active, intelligent woman, came to St. Thomas's Hospital on April 4th, 1887, and gave the following account of her symptoms. On the evening of March 31st she had a severe and prolonged fit of sneezing, and immediately afterwards noticed a spot before the right eye, which has persisted with scarcely any appreciable change. It is in the centre of the sight, and moves only with movements of the eye. It appears dark, almost black, with a little claret colour at the margins.

On examination, the right eye externally appeared normal; the pupil was small and active; central vision very defective, patient could not count fingers at two feet. Peripheral vision good.

The ophthalmoscope revealed a very large dark hæmorrhage occupying the macular region, of the shape and size represented in the rough sketch (fig. 1) which I made at the time. (I think the transverse diameter of the hæmorrhage is rather too large in the drawing; the vertical measurement is about right.) The retinal vessels above and below are hidden in part of their course by the extravasated blood; where visible they exhibit no unusual appearances. Fundus elsewhere healthy. Media clear. Tension normal. Left eye normal in all respects.

Dr. Hector Mackenzie, at that time our Resident Assistant Physician, kindly examined the patient for me, and reported that there was no cardiac disease and no albuminuria. The patient was thin but healthy looking, and did not appear prematurely old. She had had no recent illness or privation.

On the 6th April the vision of the right eye had improved, so that patient counted fingers accurately at two feet, in the centre of the field. The ophthalmoscopic appearances had undergone very striking alterations, which are crudely represented in the sketch (fig. 2), made by myself at the time of examination. The large blood-clot had become divided into an upper and lower portion, separated by a narrow horizontal area with roughly parallel sides. In this interspace (fig. 2) the choroid could be well seen, but presented a curious deep red colour, as if there were a thin layer of blood-clot on the retina through which the deeper structures were plainly visible.

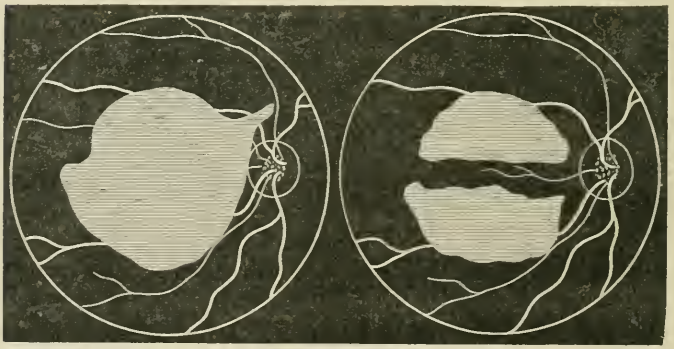

Fig. 1. Fig. 2.

A small retinal vessel had come into view running directly outwards from the optic disc. The nasal edge of the extravasation had moved slightly away from the margin of the papilla, and the narrow upward extension shown in fig. 1 had entirely disappeared. In the upper part of the hæmorrhage a few tiny bright spots were seen.

The patient did not return till April 15th, when she came saying the sight had that day become "more dull." On examination the vitreous was found to be full of blood —no red reflex could be obtained from any part of the fundus. Tension was normal; there was no pain; no cause for this fresh extravasation was ascertained.

The patient attended at intervals of one to two weeks till July 22nd. During this time the blood in the vitreous slowly underwent absorption, and vision steadily improved. On July 8th the conditions were noted, as follows:—

R. V. = $\frac{6}{18}$, + 3 D = J. 16, not J. 14.

Ophthal.—The blood-clot is chiefly in lower part of vitreous. The upper part of retina and the optic disc can be dimly seen, but no details can be made out.

After July 22nd the patient disappeared, although efforts were made to induce her to continue attending as an out-patient.

I saw her again on April 4th, 1892, exactly five years after her first visit, and the note made runs thus:—

R. V. = $\frac{6}{12}$ partly; L. V. = $\frac{6}{12}$ correctly.

R. + 4 D, reads J. 1 slowly.

L. + 4 D, reads J. 1 readily.

On ophthalmoscopic examination (with undilated pupil) I could detect no opacity in the vitreous, and beyond a slightly speckled appearance in the macular region, no changes in the fundus of the right eye. There were a few cortical striæ in the lens. The left eye, as on previous examinations, appeared normal. The patient considered that the sight of her right eye was "as good as it ever had been."

A considerable number of cases have been reported in recent years, in which large blood extravasations have occurred in the macular region of the eye, and generally circular or semi-circular in shape. As to the exact position of these hæmorrhages, opinion has wavered a good deal; they have been called in turn retinal, subretinal, choroidal, and lastly, and probably correctly, subhyaloid. There seems little doubt that the bleeding takes place from one of the large retinal vessels, and forcing its way through the anterior limiting membrane, spreads out between this and the hyaloid. It is as yet uncertain whether the blood is derived from arteries or veins, although from the turgid condition of veins described in some of the reported

cases, the probabilities are in favour of a venous origin. In my case, the prolonged fit of sneezing, causing temporary venous obstruction, lends support to this; without doubt the bleeding was an immediate result of the sneezing.

Extensive hæmorrhage into the vitreous humour, such as occurred in my patient, does not appear to be usual in these cases, judging from published records. I have not myself met with a similar case. The long interval (sixteen days) which elapsed between the first hæmorrhage and that into the vitreous suggests that possibly the source of the blood was different on the two occasions. Complete recovery from so extensive an intra-ocular hæmorrhage, in a woman between 55 and 60 years of age, is somewhat remarkable. I had no expectation, when I found the vitreous full of blood, that entire absorption would take place, leaving a practically undamaged eye. Indeed at the time I gave a very guarded prognosis as to the recovery of useful vision.

HIRSCHBERG (Berlin). Introduction to Ophthalmic Surgery. Part I.*

Prof. Hirschberg's book will be studied by specialists with interest, as it gives the author's views and practice in the most pronounced fashion, and differs both in style and arrangement from the ordinary treatises on the subject.

Ophthalmic surgery is treated of under three heads: (1) curative agents, including local and general therapeutics, operations, and dietetics; (2) the physiological eye; and (3) the diseases of the eye. The author plunges at once

* Notice of this work has been accidentally delayed.

into the subject matter of head No. 1, treating first of germicidal agents. Among them he gives the palm to corrosive sublimate solution (1 to 5,000), which he employs as a disinfectant in cataract operations. Next to this in importance he places chlorine water. Carbolic acid is only good for instruments, and worthless for wounds. Iodoform is used in cataracts complicated with dacryocystitis, or after exenteration of the orbit, or operations when there is diseased bone. The author prides himself on treating of those remedial agents only which are actually indispensable, and characteristically dismisses all other germicidal agents from consideration—with the exception of the actual cautery, of whose germicidal powers there can be no question. Iodoform, indeed, finds another use in the form of ointment to prevent skin flaps from adhering to dressings, or as an application to the nasal mucous membrane, or to the conjunctiva of scrofulous children.

Astringents are divided into the solutions of metallic salts, powders and ointments. First in importance comes sulphate of zinc, in solutions of from 1 per 100 to 1 per 1,000. Sulphate of copper and lapis divinus are nearly as effective as the sulphate of zinc, but acetate of lead is rejected in favour of diluted chlorine water. Nitrate of silver is highly commended as an agent in conjunctival affections, being used in solutions of from $\frac{1}{2}$ to 2 per 100. Prof. Hirschberg, we see, still follows the time-honoured custom of washing off the silver with saturated salt solution. The reviewer has found the application of vaseline or any oily substance a far superior method—it arrests the action of the caustic painlessly and at once. Hirschberg prefers —and therein we are quite in accord with him—sulphate of copper to solid nitrate of silver. For astringent ointment, sulphate of copper combined with cocaine and glycerine ointment, and the yellow oxide of mercury in vaseline, are recommended. Oxide of zinc and calomel are the only astringent powders mentioned.

The more purely antiphlogistic remedies meet with scant approval. Venesection is useless; leeches only useful to relieve pain; the popular artificial leech has only one posi-

tively certain effect—viz., the cicatrix on the patient's temple. Dry cupping and foot baths serve only to employ, distract or amuse our patients. The only antiphlogistic drugs recommended are pilocarpine and salicylate of soda.

There are no specific remedies for eye disease, but of course mercury and iodide of potash are treated of in connection with syphilitic eye affections. Prof. Hirschberg prefers mercurial inunction to all other forms of administering the drug, but recommends also perchloride of mercury given internally. The subconjunctival injections so popular in France are not to be recommended.

Considerable space is given to narcotics, chloral, morphia, sulphonal, &c. The latter is a most useful drug. Cocaine and atropine are also treated of, and we notice that atropine irritation (atropine conjunctivitis) does not occur in Prof. Hirschberg's practice, so that he disbelieves in the idiosyncracy which some persons are held to possess with regard to the drug. He has never seen a case of the kind, because his solutions are chemically pure and effectively sterilised. Hyoscin is recommended as a more active, and homatropine as a milder mydriatic, and eserine and pilocarpine are used in glaucoma.

After the above review of the oculists' pharmacopœia the subject of operations is taken up. Some sixteen pages are devoted to the treatment of wounds, the most perfect asepticism being insisted upon. The locality, the patient, the operator and his assistant, the instruments, dressings, and collyria must all be kept perfectly free from germs. The theatre is reserved for non-contagious cases only, is to be washed out daily after operations are performed, and again in the early morning. The couch (made of glass and iron), as well as all instruments and trays, are to be washed with strong corrosive sublimate solution (1 to 1,000). The patient gets a warm bath the evening before, special attention being paid to the hair of the head and face, and the eye is washed out with weak chlorine water for half an hour both then and the next morning. Immediately before the operation the lids and conjunctiva are well washed

with sterilised wool soaked in corrosive sublimate solution (1 to 5,000).

The surgeon and his assistants wash their hands and nails with sterilised brushes, and disinfect them with alcohol and corrosive sublimate solution (1 to 2,000), and then clothe themselves in clean linen.

The instruments are disinfected by boiling water, and then placed in 2½ per cent. sterilised carbolic solution. All dressings and bandages are disinfected by steam, Prof. Hirschberg trusting in the asepticism of no manufactured article. Four separate sterilising apparatus are used in his operating theatre. All collyria—cocaine, atropine, &c.—are also rendered sterile by steam, and a separate bottle of whatever is used is given to each cataract case. The dressing employed in cataract cases is corrosive sublimate solution. Failure of sight must be due to some lesion of (1) the dioptric media; (2) the retina; (3) the optic nerve; or (4) the cerebral nerve fibres and ganglia. The rest of the first part of the book is devoted to the subject of refraction, which is treated of at great length with all the necessary mathematical optics. It appears that Prof. Hirschberg prefers the direct ophthalmoscopic examination to all other objective tests of refraction; but this subject is to be dealt with in a later part of the treatise. It is neither possible nor desirable, in the necessarily brief space at our command, to enter upon the mathematical optics to which the remainder of this part of the work is devoted. We can heartily recommend those interested in such subjects to the original.

J. B. S.

W. A. TURNER (London). On the Diagnostic Value of the Loss of the Pupillary Light Reaction, with a note on the Oculo-facial Muscular Group. *Royal London Ophthalmic Hospital Reports, Dec.*, 1892.

As its title implies, this paper is divided into two parts, the first of which is taken up with a discussion of observations in connection with the loss of the pupillary light reaction. The observations are partly based on the clinical examination of certain cases, of which brief notes are given.

These (fifteen) cases are divided into the following groups: (1) those in which the Argyll-Robertson pupil occurred without loss of knee jerks; (2) those in which the Argyll-Robertson pupil was present on one side only; (*a*) without other nervous symptoms, (*b*) with symptoms of mixed sclerosis, (*c*) in general paralysis of the insane; (3) those in which the Argyll-Robertson pupil was associated with atrophy of the optic nerve, but without loss of knee jerks. In all the presence or absence is noted (1) of any paralysis of oculo-motor nerves; (2) of any changes in the optic nerve; (3) of knee jerks; (4) of other pertinent facts.

Certain conclusions are drawn from the facts recorded. Thus, from the fact that in one case there was a history of former diplopia, and that in two others he observed paralysis of the third nerve, which passed off, but which left behind it Argyll-Robertson pupils, the author concludes that the pupillary condition was due to a different ("central") lesion, while "the paralysis of the external ocular muscles was due either to a concomitant peripheral palsy, or was of the nature of the temporary palsies, probably of central origin, which are met with in tabes dorsalis."

In all the cases of which he gives notes, symptoms were found, more or less unequivocal, of nervous disease, except in three; of two out of these three Dr. Turner possesses

only very scanty notes dating some years back; of the third, syphilis was probably the direct cause. In the third series of cases the atrophy of the optic nerves was not so far advanced as to be itself the cause of the loss of reflex action, in the opinion of the author, so that the two conditions were probably due to one cause, viz., "a degenerative lesion common to the optic nerves and tracts, and the central mechanisms ventral to the corpora quadrigemina, which subserve the reflex contraction of the pupil." In one case an area of partial anæsthesia was found in the region of distribution of the fifth nerve; this the writer considers to be "suggestive of the presence of a sclerotic lesion, either in the trunk of the nerve or in the distribution of its roots . . . . in the medulla and pons Varolii. I am inclined to think that the cause of this will be found in connection with the distribution of the so-called 'ascending root' of the fifth nerve, rather than in the trunk of the nerve itself." It is noted that Ross regarded the ascending root of the fifth as analogous to the posterior root zone of the spinal nerves; in which area, as is well known, the lesion characteristic of tabes dorsalis is liable to be found.

The conclusion arrived at by Dr. Turner from his study of these matters is that the existence of the Argyll-Robertson pupil is highly suggestive of an early progressive change in the upper portion of the central nervous apparatus. Cases of unilateral reflex iridoplegia are useful in assisting to determine the precise seat. "In order to permit of consensual action the afferent and efferent routes of opposite sides are commissurally connected; and this occurs in two ways, first, by the passage of fibres across the middle plane from one nucleus to the other; and secondly, by fibres which pass dorsally to the aqueduct of Sylvius in the ventral portion of the posterior commissure from the proximal end of the optic tract on one side to the oculo-motor nucleus of the opposite side." (Darkschewitsch).

A lesion which blocks the direct fibres from the optic tract to the third nerve nucleus of the same side will pro-

duce loss of direct pupillary action to light, but not of consensual; to abrogate both reactions on one side, the lesion must block both the direct and the crossed fibres—must be in the sphere of the centre for the sphincter iridis. Dr. Turner does not agree with the usual text-book explanation of the occurrence of spinal myosis with the Argyll-Robertson pupil, viz., that there are two seats of disease—one causing interference with cilio-spinal fibres in the cervical region of the cord, the other in Meynert's fibres. In cases in which there are no other evidences of sclerosis of the posterior columns, he prefers to assume a single lesion as the cause of both, "the only possible situation of which is the Sylvian gray matter forming the forepart of the oculo-motor nucleus." Gowers' observation that in such cases the pupil does not dilate on stimulation of the skin is regarded as helping to confirm this view.

After giving a brief account of a few more cases, more miscellaneous, the author concludes by "suggesting the following subdivision of the fore part of the oculo-motor nucleus:

"1. A sphincter-inhibitory centre, closely associated with a sphincter-contracting centre, which two subserve the pupillary light reflex.

"2. A centre for accommodation.

"3. A centre for contraction of the pupil with convergence, in close association with the centre for the associated action of the internal recti muscles."

The second part of the article is occupied with an examination of Mendel's hypothesis that the oculo-facial group of muscles (viz., the eye-muscles proper, with the levator palpebræ, the orbicularis, and the occipito-frontalis) are innervated from the oculo-motor nucleus. The writer finds clinical confirmation of this theory in the facts that in the ordinary form of bulbar paralysis, and in hemiplegia, the orbicularis and occipito-frontalis are spared; and anatomical and pathological evidence in particular from a case of his own in which the *post-mortem* appearances confirmed the clinical opinion.

The paper of which the above is an outline is a very thoughtful one, and is well calculated to stimulate reflection upon an important subject. The author's desire to follow the safe guide which tells us to reconcile all symtoms to a single lesion, when possible, rather than to two or more, has led him to select a new site for that causing reflex iridoplegia, at all events in some cases. His objection to the usually accepted explanation is that it involves "either a double lesion or an extensive area of degeneration." But is not that just what does exist in locomotor ataxia—the great cause of the Argyll-Robertson pupil? Do we not see optic atrophy, for example, occurring early in the disease, apparently without any actual connection with the lesion of the posterior columns? Does not Dr. Turner invite us to suppose a lesion of the ascending root of the fifth nerve in the case of partial anæsthesia above referred to,—a scattered lesion, therefore? And while it is quite true that in some of the cases of locomotor ataxy, &c., there are no other symptoms pointing to the posterior columns, is that not simply because we are able very early to recognise this disturbance of a nerve-conduction mechanism—a mechanism which perhaps is easily upset? One symptom does point in this direction, but this Dr. Turner somewhat arbitrarily disallows, viz., the loss of dilatation of the pupil on cutaneous stimulation—the loss of what we might call (if we may coin a word) "pain-mydriasis." Besides this, a lesion of the first sub-division of the (*i.e.*, Dr. Turner's) oculo-motor nucleus would, either on the one hand, not produce myosis, or on the other would fail to cause loss of light-reflex, pure and simple. For if it produced myosis at all, that could only be by each successive contraction to light remaining stationary even after the removal of the stimulus. While this condition is in process of being established there must manifestly be presence of the light-reflex, or this myosis could not be brought about, and when it is established we should have a pupil contracted ad minimum, *i.e.*, incapable of contracting further on accommodation—a condition, namely, which is not the Argyll-Robertson pupil. One point which would help in

the elucidation of this subject is the knowledge of whether the myosis precedes or occurs at the same time as the loss of light-reflex; if there is any difference in time of onset, this would help to indicate the existence of a double lesion; but where so many factors enter into the calculation (such as complexion, age, habit of body, refraction, &c.), this evidence would be very difficult to obtain; in addition to which there is the difficulty of comparing the movements of a small, with those of a medium, pupil, in response to light and other stimuli.

W. G. Sym.

GOLDZIEHER (Budapesth). A Case of Tubercular Tumour in the Corpora Quadrigemina, with Bilateral Ophthalmoplegia. *Centralblatt für Augenheilkunde, Feb.*, 1893.

A. P., aged 5, was brought to Goldzieher first on March 30th, 1891; the child was of healthy parentage, and till a short time before he was seen had been strong and well. In the end of January the patient suffered from a high fever, with swollen face and toothache, the illness lasting eight days. From that time, the child lost flesh, the gait became uncertain at first, and then more tottering; he got steadily weaker, although the appetite remained good; there was great somnolence, the boy falling asleep whenever left to himself. The condition had got worse under electrical treatment.

At date of examination the child was pale and apathetic; there was almost complete bilateral ptosis, the eyes deviated outwards, and all the muscles supplied by the third nerves were paralysed. The pupils acted normally; accommodation appeared to be active, for the child easily

recognised objects held quite close; the fundus was normal; other cerebral nerves seemed to be unaffected; sensation and tendon-reflex were as usual. Weakness of the lower limbs and staggering gait were very marked. Intelligence did not appear to have suffered; the patient was very apathetic, yawned incessantly, spoke with difficulty, and seemed as if just wakened from sleep. When one did get his attention attracted, his answers, though of the shortest, were quite intelligent. Such was the condition up to April, 1891, when he was shown to one of the medical societies of Budapesth. But gradually the somnolent condition passed off, and in its place came a peculiar restlessness, at times amounting to acute mania, to be succeeded again by apathy. Sometimes the child started in his sleep and uttered dreadful shrieks. Weakness increased, so that he could no longer walk.

On April 29th, the ocular condition was much as it had been; pupils reacted promptly; the right arm was flexed and spastically contracted. This spasmodic flexion sometimes relaxed a little, but never entirely ceased. Sudden, violent, spasmodic movements of the limbs and of the whole body took place at intervals, so that precautions had to be taken against the child being thrown out of bed. These convulsions were apt to come on if he was thwarted or annoyed; voluntary movements also were liable to set them up. He became extremely irritable, and would strike and bite, and throw himself out of bed, so that he had to be tied down. Intelligence was not destroyed; he could give correct enough answers, but speech was slow, laboured and interrupted. He cried out frequently during the night; the somnolence was gone; apathy and convulsions alternated. Pulse 94, irregular. Matters went on growing worse, with opisthotonos, twitching of facial muscles, incoherence, &c., till he died on May 23rd (?) *Post-mortem*, there were a few small hæmorrhages in the centrum-ovale; the lateral ventricles were somewhat enlarged, and contained clear yellow serum. Corpora quadrigemina enlarged, their surfaces injected. Just under these surfaces, and pushing

against the upper dorsal part of the pons, was found a tumour the size of a hazel-nut and well circumscribed, rough in surface, firm in substance, and grayish pink in colour, with a cheesy centre. There was moderate softening round about. The aqueduct of Sylvius was narrowed by the pressure; the central gray matter of the third and fourth ventricles showed no obvious change (naked eye). The tumour proved to be tubercular.

From the above case Goldzieher draws the following conclusions. (1) A tumour occupying the whole area of the corpora quadrigemina need not produce blindness. (2) Complete ophthalmoplegia externa need not be associated with primary changes in third nerve nuclei. Probably in this case there were no hæmorrhages in the nuclear area. (3) Ophthalmoplegia, combined with staggering and inco-ordination of the lower limbs, points to a lesion of the corpora quadrigemina. The sleepiness and stumbling gait as of a person newly awaked, which were observed in the earlier part of his patient's history, and which passed off to be succeeded by apathy and convulsions, seems to have arisen from the hyperæmia in the nuclear area while the tubercular nodule was forming. (4) Convulsive twitchings of the extremities, with general epileptiform convulsions, can be caused by a lesion of the corpora quadrigemina alone. In other cases, such as Nothnagel's, these convulsions have been ascribed to meningitis, cerebral hyperæmia, or increased intracranial tension; but in this case there was certainly no meningitis, and probably no hyperæmia or increased tension inside the cranium. As bearing upon this, it is also to be observed that there was no appearance of choked disc or optic neuritis. The convulsions must therefore be looked upon as caused simply by the irritation of the corpora quadrigemina.

Reference is next made by Goldzieher to Ferrier's experiments on monkeys, in which he obtained convulsive movements of tail and lower extremities, trismus and opisthotonos, on stimulation of the corpora quadrigemina, and in particular to the fact that a peculiar cry was uttered only but invariably when the posterior pair were

stimulated. Goldzieher's patient was observed to utter just such a cry, which was described as resembling the cry of a beast (thierisch).

W. G. Sym.

E. Meyer (Paris). Note on a Particular Form of Infectious Keratitis. *Revue générale d'ophtal., Jan.*, 1893.

Meyer describes a particular form of infectious keratitis, of which he has seen four examples.

The inflammatory process first shows itself as a small round spot at the centre of the cornea. This spot, which is absolutely circular, lies beneath Bowman's membrane, in the anterior layers of the cornea. It increases in size, but invariably preserves its circular form, and does not involve the conjunctiva. There is no secretion of tears, and the unaffected parts of the cornea retain their transparency. The patient experiences no pain, and the subjective symptoms consist of slight photophobia and impaired sight, due to the central position of the lesion.

As soon as the central spot has attained a diameter of 3 to 4 mm. it ceases to enlarge, and becomes surrounded by a grayish, semi-transparent halo. It may remain stationary in this condition for some weeks, without any alteration, either interstitial or epithelial, in the substance of the cornea. Sooner or later it may, however, resume its march, renewed growth then manifesting itself as a circular white band around the gray halo. To recapitulate, the clinical picture of this form of keratitis, when fully developed, is as follows. First, a round central spot; secondly, an intermediate halo of grayish colour; and lastly, a peripheral white fillet.

The retrogressive stage of the keratitis is marked in the first instance by disappearance of the peripheral band, the halo next clears up, and finally, the central white spot undergoes absorption. The last statement requires, however, some qualification; for the author remarks that traces of the central opacity may be detected by careful examination for long after the apparent termination of the disease.

Meyer's youngest patient was 16; his oldest, over 60. It is of great importance to note that in every case the keratitis followed debilitating illness. For instance, No. 1 was convalescent from influenza; No. 2 had recently suffered from prolonged fever; and Nos. 3 and 4 had been the subject of herpes frontalis.

With regard to treatment, the author declares that the remedies in common use are without effect in shortening the course of the disease. He recommends the employment of weak sublimate solutions, and is of opinion, further, that the galvano-cautery or syndectomy is indicated in some cases.

SYDNEY STEPHENSON.

A. A. HUBBELL (Buffalo). Congenital Ptosis with Synchronous movements of the Affected Lid and Lower Jaw. *Archives of Ophthal., xxii.* 1.*

T. W. B., aged 7, is a well-developed, healthy, and unusually intelligent boy. He is one of a family of three children, whose parents are strong and well. There is no history of malformation either on the father's or mother's side. The head is well-shaped, and the face, except for right-sided ptosis, is symmetrical.

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* Compare with short article on the same subject by W. N. Sinclair, *Oph. Rev.*, March, 1893, p. 80. [ED.

It was noticed shortly after birth, that the right eyelids did not open with the left, but remained closed, opening only synchronously with the mouth, and closing again so soon as the mouth was shut. This condition has remained without apparent alteration up to the present time.

Careful examination by the author showed that with the mouth closed there is not the slightest movement of the right upper lid when the left lids are opened to the ordinary extent, or the left eye-ball is directed upwards, or when the eye, head, or lower jaw is turned to one side. If still with the mouth closed, the boy is told to open both eyes as widely as he can, the right upper lid is raised a little, but this is due to the action of the occipito-frontalis alone, and not to that of the levator palpebræ. When, however, the mouth is opened the right upper lid at once rises involuntarily and unconsciously. The greater the depression of the lower jaw, the higher is the upper lid lifted, *i.e.*, the right levator acts synchronously with the digastric, and the degree of contraction of the former is directly proportionate to that of the latter. But it is to be noted that the orbicularis, whose function is normal on both sides, can overcome this associated movement of the eyelids with the jaw, seeing that the right eye can be voluntarily kept closed while the mouth is opened.

The right globe is of the same size as the left, the iris healthy and the pupil active to light. There is marked divergent strabismus, with a slight amount of downward deviation. The globe cannot be turned upwards beyond the horizontal meridian, or inwards for more than a very short distance beyond the vertical median plane, but the movements downwards and outwards are quite as extensive, if not more extensive, than usual. Refraction of both eyes is emmetropic, and the fundus of each is healthy, but the vision of the right is defective—fingers at four feet—while that of the left is normal.

In no recorded instance of this affection has there been a *post-mortem* examination made, so that the explanation is at the best, somewhat conjectural. The author accepts the view which is more or less generally adopted that

there has been some interchange between the nucleus of the descending motor root of the fifth nerve and the nucleus of the root of the third, and that probably combined with this there has been a lack of proper development of the nucleus of origin of more or less fibres of the third nerve. In his own case he believes that "the origin of the fibres supplying the levator muscle has been transferred from the nucleus of the third nerve to that part of the nucleus of the fifth which sends fibres to the anterior belly of the digastric muscle. The nucleus of the third nerve seems to be undeveloped in that portion from which arise the fibres supplying the levator of the upper lid, the superior rectus, and the internal rectus muscles, while that part sending fibres to the inferior rectus (possibly the inferior oblique) and the iris is normally present. Thus we get in this case the ptosis, the divergent strabismus, the downward deviation and the associated movements of the upper lid with the lower jaw."

N. M. ML.

F. C. Hotz (Chicago). A remarkable Case of Complete Paralysis of the Internus and Externus of each Eye.—*Archives of Ophthal. xxii.*, 1.

This case is so unusual, both as regards the actual condition and the course of the disease, that it is worth recording.

G. F., a druggist, age 22, first consulted the author on May 3rd, 1892. He had had pneumonia two years previously, and five years ago was seriously ill for a fortnight from "heat-stroke," but in both instances he made a complete recovery, and had been in thoroughly good health since. The patient is a man of regular and

temperate habits; he denied all possibility of syphilis, and the writer had no reason to disbelieve him.

Two months before he was seen by Hotz, while compounding some medicine, he suddenly found that if he wished to look from one object to another he had to turn his head instead of his eyes. This condition made him giddy in walking, and was accompanied by a persistent dull headache. It remained, so far as he could judge, just as it was when first noticed, neither improving nor getting worse.

There was complete paralysis of both external and internal recti. The left eye was exactly in median position; the right showed a convergence of ten degrees (by the perimeter). In neither was there the faintest trace of any lateral movement. Upward and downward movements could be freely made, but they were strictly limited to the perpendicular line, no deviation to the side being observable. The eyes were both emmetropic, could read J.1 up to 5 inches; the pupils reacted readily to light, and the fundus was normal in each. The test for diplopia gave the following result:—In the middle line, and to the left of it, the patient saw two images (homonymous), but as soon as the light crossed the median line to the right, the diplopia entirely ceased.

On May the 10th the case was exhibited at the American Ophthalmological Society; the paralysis of internal and external recti was still complete, but there was also a complete paralysis of the right side of the face. The members of the Society who were present convinced themselves as to the accuracy of the author's observation.

The patient was not again seen till June 17th, when he reported himself to Hotz, as perfectly well. There was now no facial paralysis, and the lateral movements of the eyes were normal. The right eye, however, still showed a convergence of ten degrees. At a subsequent examination on November 30th, it was found that this convergence had disappeared, and binocular vision was fully re-established. Measurements by the perimeter gave a rotation of each eye, outwards 65°, inwards 50°.

The history of the course of the disease between May 10th and June 17th, as narrated by the patient is as follows: He had been taking iodide of potassium, while under Hotz's observation, and although it caused dyspepsia and nausea, he persisted in increasing the dose until he took twenty-seven grains three times daily. That was on May 10th, and the next morning he awoke with all the symptoms of a violent gastritis, vomiting blood, etc. He was confined to bed for a week, during which time the left side of his face and his arms became paralysed, and the legs also were so much affected that he could not walk alone. A fortnight later he began to take strychnine (gr. 1-60), and in a few days he noticed a slight improvement. Recovery was gradual but steady from this time, the paralysis disappearing first from the legs and arms, then from the face, and finally from the lateral recti muscles.

N. M. ML.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

THURSDAY, MARCH 9TH, 1893.

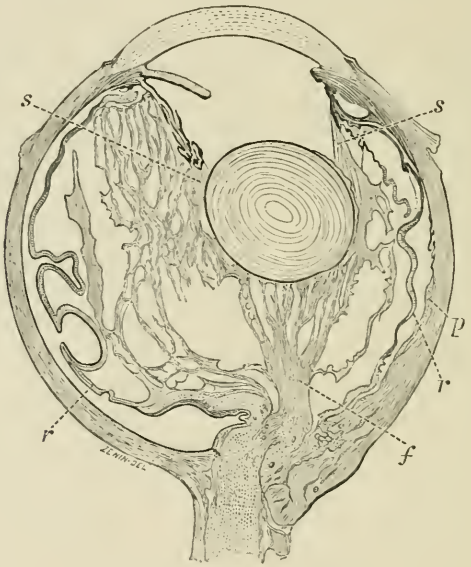
W. A. BRAILEY, M.D., in the Chair.

Before beginning the ordinary business of the Society, the Chairman, in a few well-chosen remarks, alluded to the great loss the Society had sustained in the death of Dr. James Anderson, a former Secretary and valued member of the Society.

Microphthalmos.—Mr. Treacher Collins showed a series of lantern slides, illustrative of the changes he had found in three microphthalmic globes. Two of the three eyes had been obtained from a child who died when four days old of congenital heart disease. In the left eye (see fig.) the lens was displaced backwards, being situated nearly in the centre of the globe. In the middle of the vitreous was a band (*f*) composed of elongated cells and fibres, with blood vessels amongst them; it passed from the posterior pole of the eye just below the optic disc to the back of the lens. Band of fibres and cells, similarly situated, had been described by Hess, and Mr. Collins agreed with him that they were probably the result of atypical development of the intruding mesoblast, which should have formed vitreous. The displacement of the lens was due to this band of fibres and cells having kept it moored, while the anterior portions of the eye grew forwards, the ciliary processes and fibres of the suspensory ligament becoming thereby greatly stretched. In this eye there was also a coloboma of the iris. In the right and left eye there was posteriorly below the optic nerve a protrusion due to a projection of retinal tissue through a gap in the sclerotic.

The third eye, which was removed from a lad, aged 18, had connected with it, in a similar position, two large cysts lined throughout by degenerate retina. The cysts in this case, and the projections from the eyes in the other, had probably been caused by the protrusion of one of the

folds, which are normally present in the human fœtal eye, through the ocular cleft and its sequestration in the surrounding mesoblast; a process analogous to the formation of dermoid cysts by the inclusion of a portion of cuticular



DESCRIPTION OF FIGURE.

Semi-diagrammatic section of the left eye of Case 1.

(*s*) Suspensory ligament of lens stretched and attached to elongated ciliary processes.

(*r*) Retina, much folded.

(*f*) Fibrous tissue in centre of vitreous, holding lens back.

p points to pigment epithelial layer at the position where it ceased to be pigmented and where the choroid ended.

epiblast in one of the branchial or other fœtal clefts. A theory somewhat similar to the above had been propounded by Kundrat. In none of these eyes was there any indication that the changes found had resulted from intrauterine inflammation.

Removal of a Chip of Steel from the Vitreous.—Mr. Tatham Thompson read notes of a case in which he had removed a

large fragment of steel from the vitreous by means of the electro-magnet. Two peculiar points were mentioned in connection with the case. First, the extraordinary track followed by the foreign body, which had passed through the upper lid almost at right angles to the skin surface, and, penetrating the sclerotic at 7 millimetres from the corneo-sclerotic junction, had apparently turned on its axis, as it was found to be standing vertically in the vitreous, with its long axis almost parallel with the lid surface, and its lower extremity resting on the retina embedded in lymph. The second point was the way in which the foreign body, which measured 11 millimetres by 2 millimetres, and weighed 0.037 gramme, had been tolerated, the patient not complaining of bad symptoms till a fortnight had elapsed. Three weeks after the accident the fragment was removed. A flap of tissue was raised at the outer side of the insertion of the inferior rectus tendon, an incision made in the sclerotic, and the fragment removed by means of the electro-magnet. The patient made a good recovery. Charts showing the field of vision before and after extraction, and the fragment of steel were shown.

Removal of Enlarged Lacrymal Glands.—Dr. Sandford (Cork) read notes of the case of a farmer who came under his care for enlargement of both lacrymal glands to such an extent that the right eyelids were completely and the left almost completely closed. A large smooth painless swelling could be felt in the region of the gland in each orbit. The patient was in good health, and had not had syphilis. There was some enlargement of the lymphatic glands in the neck, and hypertrophy of the lymphoid tissue in the pharynx. The lacrymal glands were removed, the left first and the right a month later. The result was restoration of the palpebral aperture on each side. Two years later the patient was in good health and had had no further ocular trouble. The growths on examination proved to be adenomata. Dr. Sandford referred to somewhat similar cases reported by Messrs. Adams Frost, Power, and Snell.

Foreign Body embedded in the Eye for Five Years: Removal.—Dr. Sandford reported this case. The patient, a lad aged 16, had been struck in the eye by a needle five years previously. On examination a portion of a rusty needle was seen, one end being in corneal tissue and the other end in the lens, passing through the iris. There were evidences of former iritis, and the lens was opaque. V = p. l. The portion of needle, which measured half an inch in length, and the lens matter were removed. The eye made a good recovery from the operation, and vision of $\frac{6}{9}$ and J. 2, with correction, was eventually obtained.

Living and Card Specimens.—Mr. Power: Case of Congenital Exophthalmos.—Mr. Lawford: Peculiar Changes in Macular Region, possibly of traumatic origin.—Mr. Silcock: Case of Conical Cornea.—Mr. Brailey: Case of Doubtful Nature, with many of the symptoms of Glaucoma.—Dr. Abercrombie: *Post-mortem* specimens from a case of Proptosis and Intracranial *bruit* (reported in Trans., vol. x., p. 248).

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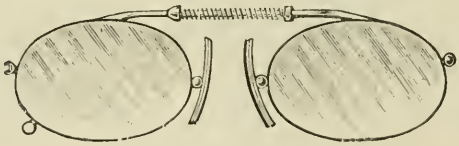

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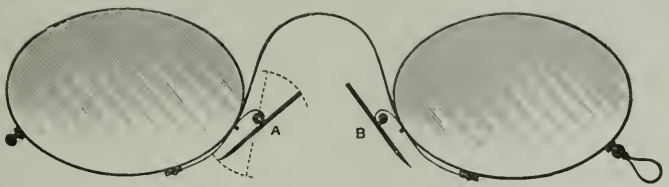

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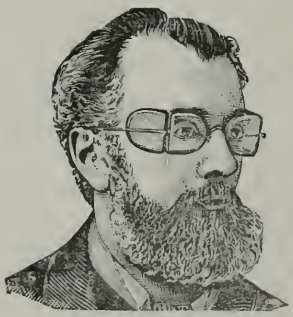

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